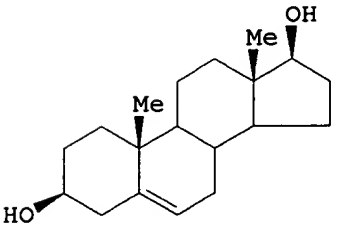
TI Adrenal dehydroepiandrosterone and human mammary **cancer**
AU Adams, John B.; Archibald, Lesley; Clarke, Christine
CS Sch. Biochem., Univ. New South Wales, Sydney, Aust.
SO Cancer Res. (1978), 38(11, Pt. 2), 4036-40
CODEN: CNREA8; ISSN: 0008-5472
DT Journal
LA English
CC 2-5 (Hormone Pharmacology)
Section cross-reference(s): 7, 14
GI



I

AB 5-Androstene-3.beta.,17.beta.-diol (I) [521-17-5] when administered s.c. to immature female rats depleted the estrogen receptor in the uterine cytosol. Similarly, dimethylbenzanthracene-induced rat mammary tumors., when incubated in vitro with 1 .mu.M I, showed translocation of the estrogen receptor from cytosol to the nucleus, as measured by exchange assays. The magnitude of depletion of cytosol estrogen receptor by I was less than that obtained with 0.3 .mu.M 17.beta.-estradiol [50-28-2], but greater than that with 1 .mu.M dihydrotestosterone [521-18-6]. Among a wide group of C19 steroids examd. as possible inhibitors of estrogen sulfotransferase [9026-06-6] both dehydroepiandrosterone [53-43-0] and I showed marked inhibitory properties. By contrast, both 7.alpha.-[53-00-9] and 7.beta.-hydroxydehydroepiandrosterone [2487-48-1] showed negligible inhibitory effects. A 7-hydroxyl group apparently modifies the ability of I to compete effectively for the estrogen receptor. Thus, the high levels of 7-hydroxylase found in human mammary tumors, and acting on both dehydroepiandrosterone and I may function in controlling the intracellular concns. of these steroids.
ST mammary tumor androgen; estrogen sulfotransferase androgen; uterus estrogen receptor androgen
IT Uterus
(estrogen receptor of, androgens effect on)
IT Neoplasm
(estrogen receptor of, of mammary gland, androgens effect on)
IT Androgens
RL: BIOL (Biological study)
(estrogen sulfotransferase inhibition by, mammary neoplasm in relation to)
IT Receptors
RL: BIOL (Biological study)
(for estrogen, of mammary gland neoplasm and uterus, androgens effect on)
IT Estrogens
RL: BIOL (Biological study)
(receptor for, of mammary neoplasm and uterus, androgens effect on)
IT Mammary gland
(neoplasm, estrogen receptor of, androgens effect on)
IT 9026-06-6
RL: PROC (Process)
(androgens inhibition of)

IT 50-28-2, biological studies
RL: BIOL (Biological study)
(estrogen receptor of mammary neoplasm in response to)
IT 53-00-9 53-41-8 53-42-9 53-43-0 58-22-0 62-83-9 63-05-8
481-29-8 521-17-5 521-18-6 739-27-5 846-46-8 1159-68-8
1232-73-1 **1963-03-7** 2487-48-1 14167-52-3 21507-41-5
63230-55-7
RL: BIOL (Biological study)
(estrogen sulfotransferase inhibition by, mammary neoplasm in relation to)

L5 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2001 ACS
AN 1978:457979 CAPLUS
DN 89:57979
TI Ultrastructural and steroidogenic characteristics of an androgen-producing
adrenocortical tumor
AU Huhtaniemi, I.; Kahri, Arvi I.; Pelkonen, R.; Salmenpera, M.; Sivula, A.; Vihko, R.
CS Dep. Clin. Chem., Univ. Oulu, Oulu, Finland
SO Clin. Endocrinol. (Oxford) (1978), 8(4), 305-14
CODEN: CLECAP; ISSN: 0300-0664
DT Journal
LA English
CC 14-10 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 2
AB A 16-yr-old woman with an adrenal cortical adenoma was studied. Clin. she
had progressive hirsutism, showed high urinary 17-oxosteroid excretion with normal plasma cortisol. Plasma C19-steroids, both unconjugated (including testosterone) and sulfate-conjugated, were greatly elevated. On ultrastructural anal. the cells in all zones of the adjoining adrenal were normal. Although the tumor cells had the general appearance of a steroid-secreting cell their structure diverged from the cells of every subzone of the cortex. This was the case particularly with mitochondria and lipid inclusions. The only endogenous unconjugated steroids detected in the adjoining cortex were corticosterone and cortisol while in tumor tissue these were present in lesser amts. The tumor tissue contained large amts. of C19-steroids, 11.beta.-hydroxy-androstenedione being quant.
most significant. An impaired defect of 21-hydroxylation in tumor cells leading steroid synthesis from corticosteroidogenesis to the C19 pathway is proposed.
ST adenoma steroidogenesis ultrastructure
IT Adenoma
(androgen-forming adrenocortical, steroidogensis and ultrastructure of)
IT Androgens
Corticosteroids, biological studies
RL: FORM (Formation, nonpreparative)
(formation of, by adrenal cortical adenoma)
IT **Cancer**
(of adrenal cortex, steroids in blood plasma and urine in)
IT Blood plasma
Urine
(steroids of, in adrenal **cancer**)
IT Adrenal cortex, neoplasm
(adenoma, androgen-forming, steroidogenesis and ultrastructure of)
IT 50-22-6 57-83-0, biological studies 63-05-8 68-96-2 80-92-2
145-13-1 382-44-5 481-29-8 521-17-5 521-18-6 901-56-4 903-67-3
1963-03-7 4150-30-5
RL: BIOL (Biological study)
(of blood plasma and adenoma, adrenocortical)
IT 58-22-0
RL: BIOL (Biological study)

(capsules, sustained-release; 5-androstene-3.beta.,17.alpha.-diol compns. for treating **cancer**)

IT Drug delivery systems
(capsules; 5-androstene-3.beta.,17.alpha.-diol compns. for treating **cancer**)

IT Bladder
(carcinoma, inhibitors; 5-Androstene-3.beta.,17.alpha.-diol as inhibitor of tumor growth)

IT Intestine, neoplasm
(colon, inhibitors; 5-Androstene-3.beta.,17.alpha.-diol as inhibitor of tumor growth)

IT Antitumor agents
(colon; 5-Androstene-3.beta.,17.alpha.-diol as inhibitor of tumor growth)

IT Drug delivery systems
(freeze-dried; 5-androstene-3.beta.,17.alpha.-diol compns. for treating **cancer**)

IT Drug delivery systems
(injections, freeze-dried; 5-androstene-3.beta.,17.alpha.-diol compns. for treating **cancer**)

IT Drug delivery systems
(injections, i.v.; 5-androstene-3.beta.,17.alpha.-diol compns. for treating **cancer**)

IT Antitumor agents
(lymphoma; 5-Androstene-3.beta.,17.alpha.-diol as inhibitor of tumor growth)

IT Antitumor agents
(mammary gland; 5-Androstene-3.beta.,17.alpha.-diol as inhibitor of tumor growth)

IT Drug delivery systems
(mucosal; 5-androstene-3.beta.,17.alpha.-diol compns. for treating **cancer**)

IT Mammary gland
(neoplasm, inhibitors; 5-Androstene-3.beta.,17.alpha.-diol as inhibitor of tumor growth)

IT Drug delivery systems
(patches; 5-androstene-3.beta.,17.alpha.-diol compns. for treating **cancer**)

IT Drug delivery systems
(solns., oral; 5-androstene-3.beta.,17.alpha.-diol compns. for treating **cancer**)

IT Drug interactions
(synergistic; synergistic interaction of 5-androstene-3.beta.,17.alpha.-diol with other antitumor agents)

IT Drug delivery systems
(topical; 5-androstene-3.beta.,17.alpha.-diol compns. for treating **cancer**)

IT **1963-03-7**, 5-Androstene-3.beta.,17.alpha.-diol **1963-03-7D**
, 5-Androstene-3.beta.,17.alpha.-diol, esters and ethers
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(5-Androstene-3.beta.,17.alpha.-diol as inhibitor of tumor growth)

IT 13311-84-7, Flutamide 84371-65-3, RU486
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(synergistic interaction of 5-androstene-3.beta.,17.alpha.-diol with other antitumor agents)

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AN 1979:16814 CAPLUS
DN 90:16814